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The Bristol Medico-Chirurgical Journal

" Scire est nescire, nisi id me
Scire alius sciret."

OCTOBER, 1924.

THE LONG FOX LECTURE:

DELIVERED IN THE UNIVERSITY OF BRISTOL ON JULY 23RD, 1924.

PROFESSOR F. FRANCIS, D.Sc., *Pro-Vice-Chancellor in the Chair.*

BY

C. LLOYD MORGAN, LL.D., D.Sc., F.R.S.,

Emeritus Professor of Psychology, Ethics and Logic, University of Bristol,

ON

THE CONDITIONED RESPONSE.

IN some interesting experiments conducted by Dr. E. J. Allen at the Plymouth Marine Biological Laboratory electric bells were suspended in the tanks in which such fishes as pollack and rudd are kept. At first there was no evidence that the fish were affected by the vibrations transmitted through the water. Food was then placed day after day in the neighbourhood of the ringing bell. After a time—a month or so—when the bell was set in vibration, with no food near, the fish assembled round it apparently in search of the customary food. Two bells,

some twelve feet apart, were fitted in the tank. By ringing them alternately the fish could be drawn to and fro in the tank towards that bell which was in vibration.

It seems, then, that the fish were stimulated by the vibrations in the water in some such way as we should speak of as hearing the bell. It seems that what we speak of as an "association" was established between the sound of the bell and the sight and taste of food. It seems that there was expectation of food when the bell was rung, and that the behaviour of the fish was guided by such expectation.

This is a conditioned response—learnt in the course of individual life. The fish, as we say, profited by "the lessons of experience." We naturally interpret this in mental terms—hearing the sound of the bell, sight and taste of food, "association of ideas," expectation, and so on. But we know very little about the mind of a fish. And in any case, such mental processes as may be present can only be surmised from that which is observed. What was observed in Dr. Allen's fish is a new manner of response in behaviour under certain conditions of external stimulation; and what may be inferred is some physiological provision for the establishment of a conditioned response, one acquired in the course of individual life.

Such new ways of response in behaviour have for long been matters of familiar observation. Young chicks peck at any small thing within pecking range with much accuracy. Let us call this a *primary response* to visual stimulation, and let us distinguish the *first occasion* on which such a primary response is in evidence. There may be first occasions in this sense on which the chick pecks at a number of different things, but on each occasion the primary response, the act of pecking, is closely similar. Now let there be, under experimental inquiry, just two different kinds of things, say, maggots and ladybirds. The primary response is to

peck at them—either of them without preference—at sight; and so far there is no observable difference in the primary response. The ladybird or maggot is seized. There is thus afforded stimulation in the mouth. The maggot is swallowed; the ladybird is rejected or tossed on one side. Each is a primary response in behaviour and is observable on the first occasion. So far we have different primary responses to different modes of stimulation. But keep the chicks under observation and watch their further behaviour. On sight of another maggot the chick pecks at it, seizes and swallows it with what seems to be added zest; but on sight of another ladybird the primary response to the visual stimulation is checked, and the behaviour of rejection is carried out, though there is no stimulation in the mouth to give this as a primary response. It is a *secondary response* elicited at sight on *subsequent occasions*. Such a secondary response is now termed a "conditioned response."

It may be said that this may be quite simply explained. The chick has profited by experience. He found that the ladybird is nasty, and therefore does not peck at it again. He found that the maggot is nice, and therefore eats as many as he can get. Yes. But how, on physiological grounds, does the niceness and nastiness take effect? What is the organic provision in the structure of the chick for that which observably happens? In what manner is the secondary response conditioned?

From the physiological standpoint the emphasis is on response to stimulation. Hence, as Professor Dendy says: "It must be admitted that the entire life of any organism consists of a series of responses to stimuli which reach it from various sources." Thus all embryological development, all modes of growth, all forms of behaviour, call for interpretation on these terms. But it is probably only when a complex nervous sytem has thus been developed that

there is the requisite physiological provision for the conditioned response.

The central nervous system—spinal cord and brain—sends forth nerve-fibres to all parts of the body. Some of these fibres transmit what we may call nerve-events inwards to the central nervous system, others transmit nerve-events outwards from the central nervous system. Thus when the chick sees a ladybird nerve-events are transmitted inwards from the retina. Out there is the ladybird. May I call it a pattern of events in the external world? By it the retina is stimulated. Shall we speak of a *receptor-pattern* of events in the retinal image? There is then a pattern of nerve-events which course inwards along many nerve-fibres to the brain. The chick pecks in primary response. There is a pattern of nerve-events running outwards along many nerve-fibres to the muscles. They call into play what are termed "effectors," since their effect (in this case) is muscular contraction. There is therefore an *effector-pattern* of events, which gives rise to a pattern of muscular change producing that form of behaviour we call pecking. So we have a very complicated incoming set of patterns of events, and a very complicated outgoing set of patterns of events. And connecting them is a central pattern of events in the brain.

Now the neuronic structure of the central nervous system is such that there seems to be a labyrinth of synaptic connections between any one incoming set of events and any other outgoing set of events. But when the chick pecks in primary response the events in the brain seem to follow a quite definite route—from the receptor-pattern in the retina to the effector-pattern in those muscles that are concerned in the behaviour of pecking. It seems, therefore, that this is initially a *more permeable* route than any other route. We have, then, to reckon with a *pattern of*

permeability in the brain or central nervous system. A primary response affords evidence of an initially permeable route connecting the incoming with the outgoing sets of events.

When the ladybird is seized there is a gustatory receptor-pattern in the mouth. Nerve-events course inwards. They find in the central nervous system an initially permeable route; and sets of nerve events course outwards to give the primary response of rejection. There seems to be no initial connection between *this* primary route and *that*. It is the establishment of a secondary connection between them that characterises the conditioned response. This secondary connection, affording what is now a more permeable route in the central nervous system, is acquired—sometimes slowly as in Dr. Allen's fish, sometimes very rapidly as in the chick—but always acquired in the course of individual life.

It may still be said that it is mentally and not physiologically acquired. Let us then briefly consider the case of salivation in the dog. It is to Professor Pavlov's work in this field of inquiry that we owe the concept of the conditioned response as he named it. When a dog is fed gustatory stimulation in the mouth induces a flow of saliva. That is part of the primary response. There is also motor response in mastication and so forth, but we here concentrate attention on the response of the salivary glands. There are, however, many other primary responses in the dog; for example, pricking of the ears when there is auditory stimulation. And initially there seems to be no connection between any two of such primary responses. But let the dog receive auditory stimulation from some definite sound concurrently with gustatory stimulation from food in the mouth on a certain number of occasions. Then what happens is that the sound by itself induces a flow of saliva

(which can be measured by inserting a cannula into the salivary duct) *without any gustatory stimulation*. A secondary connection seems to be established in the course of the experiments; and this affords a route of relatively greater permeability, or, in technical terms, of lessened "synaptic resistance." And since salivation is hardly at all (if at all) under conscious control; since, too, the nature of the investigation (into which I cannot here enter) seems to preclude mental influence in the usual sense of this expression; we appear to dig down to a purely physiological interpretation of this conditional response.

Some diagrams (not pictures) may here be helpful. Let *g* be the gustatory "pattern" and *au* the auditory "pattern." And let G be the initially permeable route from *g* in the central nervous system; Au the initially permeable route from *au*. Then we have on the earlier occasions, as synchronous—

g— G —salivation.
au—Au—pricking of ears.

But on the subsequent or later occasions we have—

G —salivation.
|
au—Au

There is no gustatory stimulation, and the pricking of the ears is (as I understand) inhibited. Nerve-events run their course from *au*, through Au to G, and thus to the salivary glands.

We may now give as a general rubric for the conditioned response some such statement as this. Let two primary responses be elicited simultaneously or in close succession on several occasions, then if, sooner or later, the one response is elicited by the *other* kind of stimulation there is a conditioned response, and there is presumptive evidence

of the establishment of a secondary route of acquired permeability within the central nervous system.

It may be asked why the route between Au and G is rendered more permeable when these brain-centres are in synchrous activity. Perhaps we do not as yet fully know. But that it does become more permeable seems to be a legitimate inference from a large body of physiological facts. And, did time permit, sundry physical analogies of the increase of the dominant activity in G by some concurrent activity in other parts of an integral system might be adduced.

When the chick rejects a ladybird "at sight" without needing to experience again its presumably nasty taste, we may express the physiological conditions thus:

Sight of l.b.—sight-route—seizing (which entails taste).
Taste of l.b.—taste-route—rejection.

This occurs on the first occasion. But on some subsequent occasion the visual nerve-events are switched off on to the taste-route, and then we have—

Sight of l.b.—sight-route.
|
taste-route—rejection.

In the case of the maggot-situation we have—

Sight of m.—sight-route—seizing (which entails taste).
Taste of m.—taste-route—swallowing.

And on subsequent occasions the taste-response does not thwart but endorses the sight-response. Thus we have—

Sight of m.—sight-route } —seizing and swallowing.
Taste of m.—taste-route }

Under observation we then say that seizing and swallowing occur with "added zest."

Thus we have two types of the conditioned response: (*a*) that in which conditioning thwarts or inhibits some

primary response and leads to its *suppression;* (*b*) that in which conditioning supports, endorses or enhances some primary response and leads to its fuller and more vigorous *expression.*

There is no call to deny—nay, rather good ground for asserting—that in many of the conditioned responses of the more highly developed animals there is at least an accom paniment of conscious enjoyment. In seeking physiological foundations one need not be disregarding mental events; one is trying to render clear what nerve processes may be inferred as the correlates of incipient mental processes. And one may find here a noteworthy turning-point in evolutionary advance. To this I shall return.

I want first to bid you remember how highly differentiated is the structure of the organism and of the brain, and how very complicated is the assemblage of physiological events therein. The structure of the neuronic system may be roughly likened to a meshwork of railway lines; the current nerve-events to the trains that are running. As there are thousands of trains that are running at the same time; so too there are thousands of nerve-events, each pursuing some primary course or a conditioned course switched off at certain " points " on the structural lines of rail. We have also to bear in mind: (1) that the sense-organs afford different kinds of external receptor-patterns which may co-exist at the same time; (2) that the outcome of this or that form of behaviour generally gives a new receptor-pattern which starts a new course of nerve-events; (3) that there are not only external receptor-patterns but also internal receptor-patterns registering the activity of the muscles and glands, and the existing state of the living organism; and (4) that all the sets of events are subject to the physiological poise of the time-being, largely determined by the subtly-varying activity of the endocrine

glands. If, then, one concentrates attention on some one conditioned response, one should do so with full realisation of the vast number of concurrent physiological events.

I cannot here deal with so widely extended an assemblage of fluent changes. It may, however, be helpful to look just a little more closely into the pecking response which I have selected for illustration.

What one notices under careful observation is that pecking, even if under this head there be included the preparatory posture of the body, is seldom the first effect in responsive behaviour of a visual receptor-pattern—the situation as presented in vision. Straightway pecking only occurs when the object is at just the right distance. At a little greater distance the first obvious response is a step or two forwards till the object is within pecking range. There is thus an enchained sequence of primary responses that lead up to the act of pecking. But what is the first link in the chain? I have devoted a good deal of attention to this question, for I deem it very important. I am convinced that the first link in the chain is the primary response of a clinging of gaze to the small object, and this clinging is maintained during the step or two forward. I believe it to be a physiological response, as are all primary responses. It is seen in the human infant within the first fortnight after birth, and it affords the foundations for the gradual development of focussed vision in the child—well described by Miss Millicent Shinn in *The Biography of a Baby*.

Aversion of gaze from too brightly illuminated objects is likewise a primary response. And unquestionably, in the chick, not-clinging, which is a step towards aversion of gaze, is a determining factor in that apparent ignoring of such things as ladybirds, soldier-beetles, or cinnabar caterpillars, which comes in due course. The clinging of gaze seems to be broken by the shaking of the head, perhaps

wiping of the bill on the ground, which the primary response of rejection entails. But since not-clinging, or the fuller aversion of gaze, is not from something which is hurtful to the eye but from something that has been unpleasant to the taste, it is so far a conditioned response acquired in the course of individual life. It is a matter that requires nice and careful observation; but my impression, after prolonged inquiry, is that there may be seen in young birds an initial turning of the gaze away from that the like of which has been unpleasant. And I feel sure that it may be detected in the infant when something nasty comes into the field of vision.

Now clinging of gaze is a primary form of that which will, later on, develop into conscious attention and interest. We seem to be justified in the belief, no doubt inferential, that it is accompanied by pleasurable enjoyment. So, too, aversion of gaze seems to betoken some felt discomfort. Indeed, we commonly speak in a wide sense of "aversion" from that which we have found to be unpleasant.

We thus pass to the psychological bearing of what we have learnt with regard to the conditioned response. Let me here say that I am one of those who believe that all physiological events are accompanied by what I call enjoyment. Thus there are thousands of events all through one's body which are accompanied by that kind of enjoyment which we call "feeling fit" or the reverse. I think that *all* the physiological events that are going on inside us contribute to the enjoyment which affords the foundations on which our minds are built. I use the word "enjoyment," in company with Professor Alexander, to include both pleasurable (positive) and unpleasant (negative) modes of feeling. This seems awkward at first, but so, too, does the use, in physics, of the word "acceleration" for diminution as well as for increase of speed. One soon gets accustomed

to the use of a word in a technical sense rather different from that of common parlance. On this understanding all the physiological events that are concerned in primary responses are accompanied by enjoyment positive or negative. But in primary responses it is accompaniment only. What I seek to show is that the conditioned response introduces a new factor of conscious *guidance* towards pleasurable situations and away from situations fraught with discomfort.

Let us here recall the common-sense view that, in my illustrative examples, the chick rejects or avoids ladybirds because they are unpleasantly nasty, and eats all the maggots he can get because they are nice and pleasantly satisfy the cravings of hunger correlated with an internal "pattern" of events. I suppose common sense would speak of this as conscious guidance towards what is pleasant and away from that which is unpleasant; and I think that common sense is so far right. But may not the physiologist who deals with conditioned responses also be right? If so, we have to connect their different points of view.

When we pass from physiological events and processes to conscious events and processes we introduce the notion of *reference*. What does this mean? It means that, if we impute conscious processes to the chick, what the little bird has in its little mind as the result of being stimulated by some physical influence *from* the ladybird, and as the result of acting *on* the ladybird, is referred *to* the ladybird which it sees, pecks at, seizes, and tastes. Thus, from the physical point of view the ladybird is a centre of *influence* on the chick's body; and from the conscious point of view is a centre of *reference* from the chick's mind. We must just accept such reference as a natural fact. It is that which characterises all conscious life as we experience it in ourselves. One is doing no violence to the usage of familiar thought and speech in saying that when one receives

light-influence from a thing one also refers what one thus experiences to the object of vision.

But when the chick receives light-influence from the ladybird, and when a set of nerve-events run their course along what I called the sight-route, there is, in the conditioned response, a switching off of the nerve-events on to the taste-route. The ladybird, however, is not then and there tasted. There is no gustatory "pattern" in the mouth. We then speak of the taste-events, called up within the central nervous system, as due to *revival*. There is physiological revival of nerve-events on the taste-route, and there is conscious or felt revival of the enjoyment that accompanies these nerve-events. Three things must here be noted: first, that, judging from our own experience, the mental taste-revival is referred to the ladybird; secondly, that it takes the form of expectation of what may follow; and, thirdly, that this present expectation (for the expectation is *present* though it has prospective *reference*) is either pleasurable or unpleasant.

So far it just comes to this. What the psychologist, on his part, speaks of as expectant revival having reference to the ladybird, with negative enjoyment, the physiologist, on his part, interprets as a conditioned set of nerve-events along part at least of the taste-route.

Now comes the point to which I invite special attention. What we have on "first occasions" is, in order of sequence: (1) sight; (2) pecking; (3) tasting; (4) (*a*) swallowing (maggot), (*b*) rejecting (ladybird).

Here the fourth stage is "this" or "that"—(*a*) or (*b*)—according as there is this or that kind of gustatory "pattern." But on "subsequent occasions," when a conditioned response is established, we have a different *and partly inverted order*, namely: (1) sight; (2) taste-revival; (3) (*a*) pecking, (*b*) not-pecking; (4) (*a*) swallowing, (*b*) rejecting.

The point for emphasis is that here taste-revival is brought forward to the second place in the sequence. Whereas actual tasting *follows* pecking, taste-revival, which we may call "foretaste," *precedes* pecking or not-pecking. This act may then either be endorsed so as to lead on to swallowing, or it may be inhibited, more or less completely, and replaced by the act of rejection. And which of the two acts follows is determined by the character of the foretaste, nice or nasty as the case may be.

It is this inversion of order, entailed by the conditioned response, that introduces a quite new factor, that of conscious guidance, or that which one may call orientation of behaviour towards those situations of which the foretaste is pleasurable, and away from those situations which, in foretaste, are fraught with discomfort. It should, however, be noted that this does not imply determination of present action by the future (for the future is not yet); it implies determination of future action by present foretaste due to actual tasting in the past. Now broaden the notion of foretaste so as to include *any* kind of enjoyment, *e.g.* that of the concert for which we have secured tickets; then the suggestion is that how we act turns on the pleasant and attractive, or the unpleasant and repellent, nature of the foretaste in enjoyment.

I turn in conclusion to a different question. There has been a long controversy as to what is called the inheritance of acquired characters. Has recent inquiry into the conditioned response any pertinent bearing on this vexed question?

The exact meaning to be attached to the word "acquired" needs careful definition. The emphasis falls on some specific modification of structure impressed in some specific way on the organism in the course of its individual life. But it is often very difficult clearly to distinguish

these specific modifications and the specific way in which they are induced. In our present context, however, it seems that there is less difficulty. It seems that the secondary or connecting routes in the central nervous system —those which characterise the conditioned response—are acquired in a pretty definite sense, whereas the primary routes, thus connected, imply hereditary permeability and are not in the same sense acquired. In other words, primary responses observable on the first occasion are inherited as such ; but secondary responses are the outcome of acquired permeability. The question at issue, therefore, *in this context* is this : Is there evidence that secondary permeability acquired in one generation is inherited as primary permeability in the next generation ?

Professor Pavlov has recently published some very interesting and important results, the outcome of his observations on mice. In these mice, as in Dr. Allen's fish, an " association " was formed between feeding and the sound of an electric bell. He dealt, however, with several generations of mice. In those with which he started some 300 " lessons " were required. But in the children of these mice 100 " lessons " sufficed, in their children only 30 " lessons " were required, and in *their* children but 5. The question therefore arises : May we, on the basis of this evidence, say that a conditioned response is inherited ? I think not.

The observations seem conclusively to show that specific secondary routes are more readily established in the successive generations. They do not *yet* show that a secondary route becomes a primary route—that apart from *any* lessons, even *one* lesson, auditory stimulation is followed by this response in behaviour. The number of occasions requisite to the establishment of this conditioned response is reduced in four generations from 300 to 5 ; but it is still a conditioned

and not a primary response. In young chicks avoidance of nauseous things—such as medicated (quinine-soaked) rice-grains—requires only two or three lessons. But I have never observed any young bird that avoided at sight nauseous insects that must have been often tasted by their parents and grandparents.

Professor Pavlov's observations are of great interest and value. They open up the question how the reduction in the number of lessons is to be interpreted. But, so far, they afford no evidence that after so many generations a conditioned response is inherited as a response of the primary order.

Into the more general question of the transmission from parent to offspring of something that facilitates individual acquisition—and this seems to be increasingly probable—I cannot here enter. It must suffice to say that what we now know concerning the function of the endocrine glands throws new light on this difficult problem.

AURICULAR FAILURE.[1]

BY

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AND

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THE effect of auricular failure on the heart has to be considered from two standpoints; first, the effect of the loss of the auricular contraction, and second, the effect of the abnormal rhythm of auricular failure on the ventricular contraction.

ANATOMY AND PHYSIOLOGY.

The function of the auricles is to act as reservoirs for the ventricles. The blood which collects in them during the ventricular systole passes into the ventricles at the commencement of diastole; and as diastole goes on, first the ventricles fill and then the auricles. The auricular contraction then distends the already filled ventricles, making certain that a full amount of blood shall be pumped into the aorta and pulmonary arteries every time that the ventricles contract. Loss of the auricular contraction will, on that account, lead to an insufficient filling of the ventricles. The effect of abnormal rhythms of the auricles on the ventricular contraction will be considered later.

[1] We have to express our sincere thanks to the Colston Research Society for helping us to defray the expenses of the investigations alluded to in this paper, as well as those of the illustrations; to Dr. Geoffrey Hadfield for furnishing us with the photomicrographs; and to Messrs. John Wright and Sons for the loan of blocks from a book on *Rheumatic Heart Disease* which they are just bringing out.

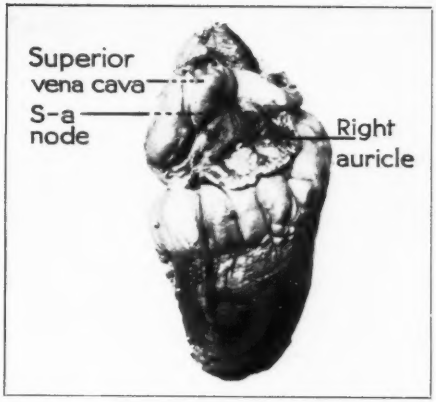


FIG. 1.

Photograph of heart, showing position of sinu-auricular node.

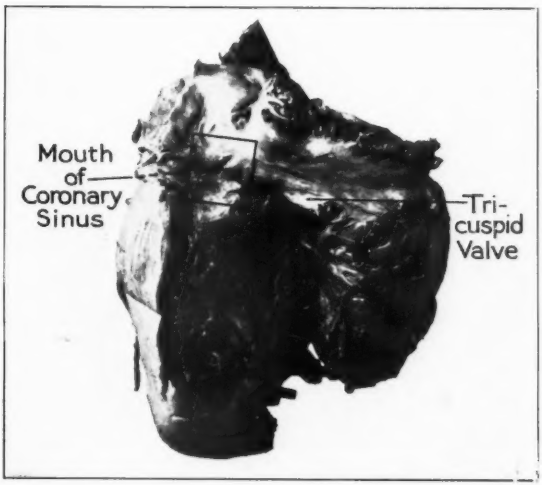
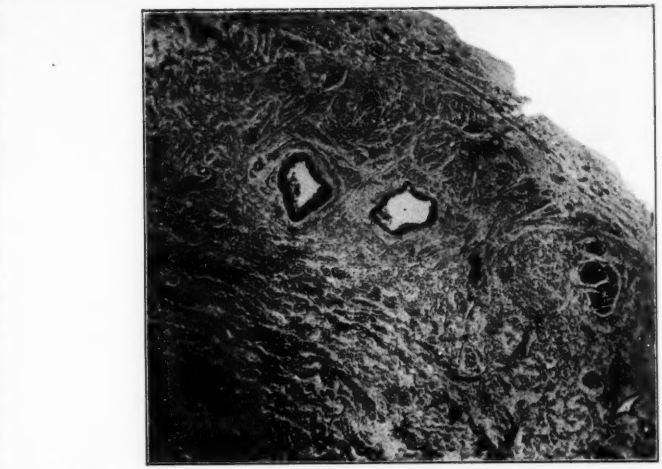
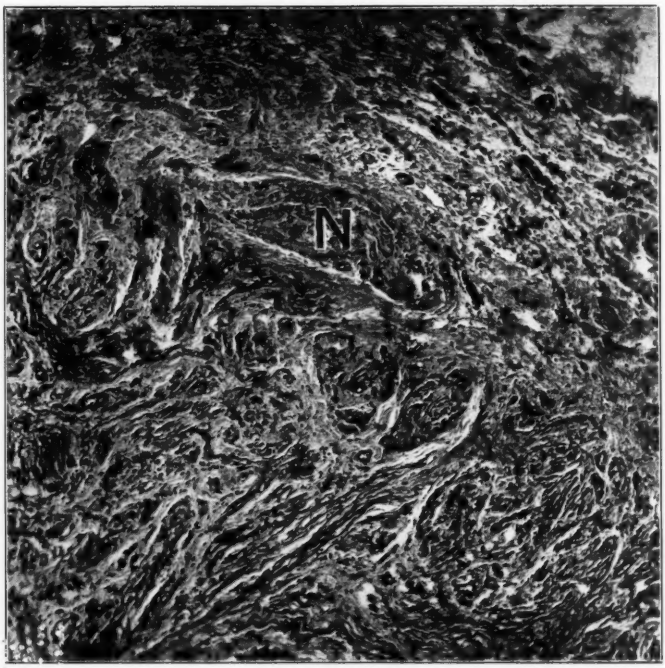


FIG. 2.

Photograph of heart with right auricle and ventricle opened to show position of auriculo-ventricular node and bundle. The rectangular outline is that of the block cut out to provide serial sections of these structures. The block is cut transversely for this purpose.





FIGS. 3 and 4.

Sinu-auricular node.

The upper (low-power) photomicrograph shows the general structure of the node. The darker area is that of the general auricular muscle; the rest is that of the node, with its central vessels. The lower (high-power) one shows a nerve trunk N sending its fibres into the substance of the node, which is mainly composed of bundles of fine muscle fibres.

The contraction of the heart is started at the upper part of the right auricle by a collection of cells known as the sinu-auricular node (Fig. 1). This node is situated in the wall of the right auricle, where the superior vena cava joins the right auricle and the base of the right auricular appendix. It is an elongated structure lying at right angles to the length of the superior vena cava, and is continued for a small distance down the sulcus terminalis.

It is composed of dense fibrous tissue in which are embedded small fine muscle fibres (Fig. 3). A large number of nerve fibres lead into it (Fig. 4). This collection of cells has the property of originating a stimulus for the contraction of the heart muscle.

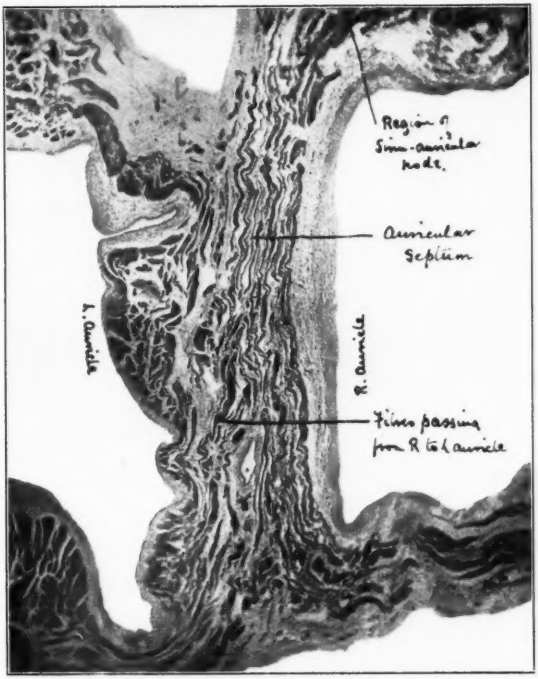
Much controversy has taken place as to the path by which this stimulus is carried on to the auriculo-ventricular node. Work has been published stating that there is a direct path from node to node, and this is said to have been proved by electrical means. Three years ago we set out to look for this path by means of serial sections of hearts. Six hearts were cut and examined, and the results of this search proved to us that there was no direct path, but that the stimulus passes to the A–V node by means of the ordinary auricular muscle, the contraction of the nodal fibres of the S–A node spreading to the auricles by direct continuity of fibres. All the muscle fibres leaving the S–A node can be traced away into the ordinary auricular musculature. There is no trace of any specialised muscle path through the auricular walls. We also found that the spread of contraction from the right to the left auricle took place by two main paths (Figs. 5 and 6). Firstly, fibres from the node passed down to the inter-auricular septum, where fibres from the right crossed the middle line and were continued round the back of the left auricle. Secondly, a broad band of fibres, passing over the tail of the right auricular appendix,

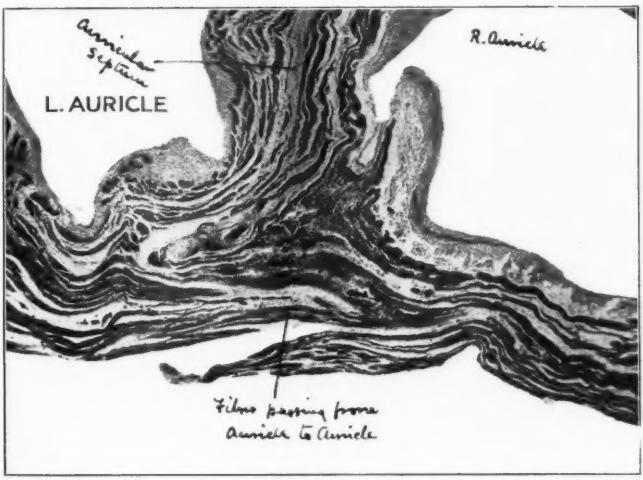
swept backwards and passed straight across to the back of the left auricle. Thus we see how the contraction starting in the right auricle causes the contraction of both chambers. The contraction wave, therefore, passes from the sinus node into the auricular muscle of both auricles, and by this means reaches the base of the inter-auricular septum, where it meets another collection of cells termed the auriculo-ventricular node. This is situated on the right surface of the septum just to the right of the opening of the coronary sinus (Fig. 2). The node is very similar to the sinus node, consisting of a network of fine muscle fibres embedded in fibrous tissue. There are many nerve fibres passing into it, and many ganglion cells can be seen amongst the muscle fibres. From this node a bundle of fibres passes across the right side of the inter-ventricular septum towards the *pars membranacea septi*. At this part it divides into two branches; the right one runs down the right side of the septum, the left branch pierces the septum and runs down its left side.

These pass down to the apex and then subdivide into innumerable branches, which are distributed to the muscle fibres of the ventricles, and are so arranged that the muscle fibres of the ventricles contract in a definite order, commencing at the apex and gradually reaching the base.

This sequence of contraction is only possible when the stimulus for contraction reaches the ventricular muscle by means of the bundle. In certain cases the stimulus originates in the ventricular muscle itself on one side or the other, and in this case the spread of contraction does not occur in this sequence, with the result that the power of the contraction suffers.

This different type of contraction evokes a particular kind of electro-cardiogram. If we look at a normal electro-cardiographic tracing (Fig. 7), we find that the waves, or





FIGS. 5 and 6.

Low-power photomicrographs exhibiting the muscular bands which join the two auricles.

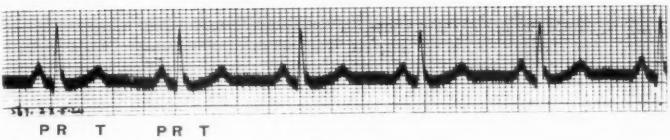


FIG. 7.
Normal electrocardiogram.

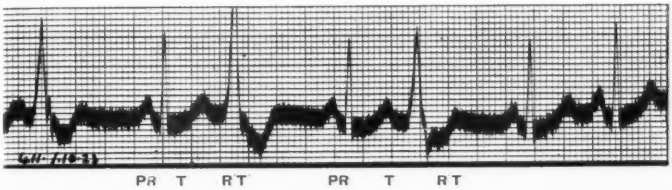


FIG. 8.
Electrocardiogram showing premature beats R'T' arising in the ventricles.

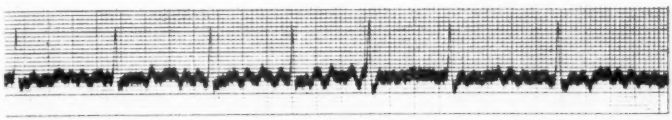


FIG. 9.
Electrocardiogram showing auricular fibrillation.

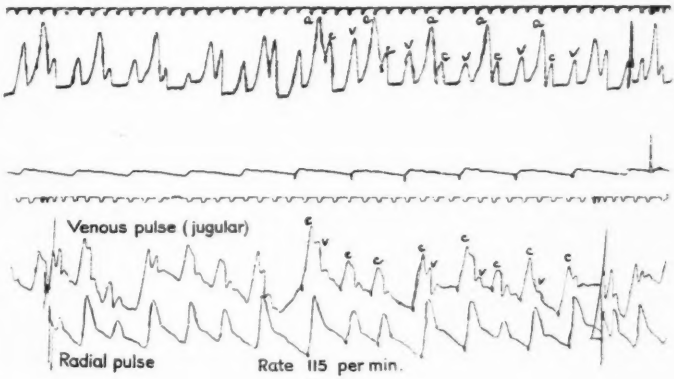


FIG. 10.
Polygrams, the upper normal, the lower from a case of auricular failure.

complexes as they are called, are of three kinds: "P," which is due to the auricular contraction and which occurs from 0.14 to 0.18 of a second before the "R"; "R" denotes the onset of ventricular contraction, and "T" represents the end of ventricular contraction; a normal R and T denotes that the ventricular contraction was called forth by a stimulus reaching the ventricle by the bundle, and is what is called a supra-ventricular complex. An abnormal ventricular contraction arising from the muscle itself gives a very different complex (see Fig. 8).

Symptom and Signs of Auricular Failure.

In a tracing from a case of auricular failure (Fig. 9), *i.e.* after the onset of auricular fibrillation, it is seen that the ventricular complexes are of the supra-ventricular type, therefore the ventricular contractions are originated by means of stimuli passing down the bundle in the normal way. It is also seen that the "P" wave is absent, but that it is replaced by small irregular waves which occur continuously, being interrupted by irregular spaced ventricular complexes. These correspond with the irregular ventricular beats that are perceptible clinically as the "totally irregular pulse." It was this kind of irregularity that first attracted attention to the syndrome of auricular failure. Mackenzie, Wenckebach and Hering noted the occurrence of a kind of arrhythmia named by them "total" or "perpetual," and associated, as they showed, with the ventricular form of venous curve, *i.e.* one from which the usual auriculo-systolic movements were absent. Lewis carried this further by showing that small irregular waves were found in the electro-cardiogram of total arrhythmia, and that these waves were caused by "auricular fibrillation."

By these successive steps the symptom-complex of auricular fibrillation has become more and more clearly defined, so that now it is easy to recognise.

There are four chief clinical manifestations of auricular failure.

The first cardinal sign is the particular kind of arrhythmia alluded to above. The characteristic fact about this irregularity is that it is complete; the normal rhythm is entirely replaced by a series of beats that vary altogether in force as well as in duration. If the pulse be counted for a number of successive periods of five seconds each, it will usually be found that each period embraces a different number of beats. It is quite different from all other kinds of arrhythmia, not only in that it is more definite, but also in its quality; since in this form of irregularity alone is there no vestige of the original rhythm left. Nearly always it is a quick pulse, less constantly so in the senile than in the rheumatic cases; and the heart rate is quicker than the pulse rate because of a number of small beats which fail to reach the wrist. This discrepancy tends to disappear under successful treatment.

When the substance of this paper was read to the Bristol Medico-Chirurgical Society this type of arrhythmia and its slowing after treatment were well illustrated by Mr. Mitchell's microphone apparatus.

The second feature of auricular failure is the disappearance of all evidence of auricular systole. The auriculo-systolic wave of the jugular pulse is missing, and the venous pulse is of the ventricular type (Fig. 10). That movement in the electro-cardiogram which is the outcome of auricular systole also disappears (Fig. 9). Finally, any presystolic bruit that may be present—as it often is, since it is in a case of late cardiac rheumatism with mitral stenosis that the auricle so frequently fails—also disappears; leaving the diastolic vibrations, if any there be, to end vaguely at an appreciable interval before the next first sound.

Thirdly, all the signs of circulatory stasis are increased.

The patient becomes more breathless, cyanosis is more pronounced and often more livid in character, the jugular veins stand out prominently, the liver becomes swollen and tender, and râles appear at the bases and edges of the lungs.

Finally, in the jugular tracing and in the electrocardiogram (see Figs. 9 and 10) evidence of a rapid irregular movement is visible, occasionally in the former and invariably in the latter.

So far as the patient is concerned, the advent of auricular failure is sometimes so insidious as to attract no attention. Nearly always, however, it brings with it a definite increment of the dyspnœa and other symptoms from which the patient is already suffering. Sometimes the onset is so sudden, and attended by so great an exacerbation of circulatory difficulty, as to cause vomiting and collapse, and to bring the patient down as if with an acute illness. At the other extreme are the persons who have no idea that the action of the heart has undergone any serious change. Sometimes it comes on at first in transient periods of failure, separated from each other by ever shortening intervals. Occasionally, and rather mysteriously, the periodic type of failure may persist for decades, the heart in the intervals appearing to behave quite normally. Pain is a rare concomitant of auricular failure, presumably because it is more especially a symptom of left ventricular insufficiency. Sometimes patients complain of palpitations, but not as often as might be expected from the rapidity of the heart's action and the frequency of the premature ventricular beats that are principal features of the syndrome.

Auricular failure is nearly always a late feature of the chronic forms of cardiac disease. Its incidence in the different types of cardiac disease may be expressed by the following table, extracted from notes on hospital in-patients and on patients recently seen in private practice :—

TABLE I.

	Hospital Patients.	Private Patients.	Total.
Rheumatic heart disease ..	48	29	77
Syphilitic heart disease ..	2	1	3
Thyroid heart disease ..	2	4	6
Ulcerative endocarditis ..	2	1	3
Cardiorenal disease (hyperpiesis)	3	15	18
Senile (decrescent) cardio-vascular disease	11	28	39
Various (alcoholism, acute infections, etc.)	4	13	17
	72	91	163

The analysis of notes from private practice displays a larger percentage of cardio-sclerotics and a smaller one of cardio-rheumatics than the hospital series, but these two remain the dominant types.

This is explained not only by the greater general incidence of those kinds of cardiac disease, but also by the particular predilection which they show for an attack on the auricular functions. The nature of that predilection will appear presently. Indeed, a glance at the nature of the pathological processes of senile heart disease shows that here, at all events, it must be myocardial degeneration that destroys the auricular efficiency. About rheumatic heart disease it is not so obvious; for in its earlier phases there is a definite and highly characteristic form of interstitial myocarditis, and it has been suggested that this, or its residuum, is responsible for upsetting the auricular function. That this is not so is suggested by the following tables, which show that auricular failure is much more often seen in the later decades of cardiac rheumatism than in the earlier phases, when the interstitial inflammation is more pronounced :—

TABLE II.

Of 58 cases of cardiac rheumatism with total arrhythmia				
2	occurred	in	patients	aged 10 or less.
2	,,	,,	,,	11–20
8	,,	,,	,,	21–30
14	,,	,,	,,	31–40
17	,,	,,	,,	41–50
15	,,	,,	,,	51 upwards.

TABLE III.

Age.	Cases of rheumatic heart disease studied by graphic methods.	Total arrhythmia in. Number.	Per cent.
Up to 10	22	1	4.5
11–20	89	1	1.1
21–30	45	3	6.6
31–40	35	5	14.2
41 onwards ..	25	12	48.0

MORBID ANATOMY.

It was in order to complete a study of rheumatic heart disease, also to satisfy ourselves on the point whether auricular failure is indeed due to degenerative processes, and particularly to find out whether such processes caused failure by a general attack on the auricular muscle or a special attack on the sinu-auricular node, that we undertook an inquiry into the histopathology of auricular failure. To this end a number of hearts from persons dying of cardiac disease with auricular failure was examined. Of these, ten were fully and eight partially examined. The accompanying table shows, among other data, what were the forms of cardiac disease in each case. In addition, a series of twenty-six hearts from persons dying of cardiac disease without auricular failure was also examined fully. The

TABLE IV.

CASES IN WHICH THE SIGNS OF AURICULAR FAILURE WERE NOTED DURING LIFE.

(*a*) COMPLETE EXAMINATIONS.

Ref No.	General Features of Case.	Duration of Failure.	Sinu-Auricular Node.	Auricular Muscle.	Ventricular Muscle.	A-V Connections.	Remarks.
70	F., 20. Malignant endocarditis supervening on chronic cardiac rheumatism.	A few days.	No abnormality.	Slight cloudy swelling and fatty change.	Slight cloudy swelling and fatty change.	Nil except usual vascular changes of chronic cardiac rheumatism in neighbourhood of central fibrous body.	No active inflammation of myocardium
74	M., 30. Chronic cardiac rheumatism with mitral stenosis, etc.	Months.	Large hemorrhage into middle of node; about a quarter of its length affected; at the worst point the node was almost cut in two. The blood appeared to come from the neighbouring part of the right auricle. Much of the clot was organised and had been there some time. About one-fifth of the total nodal tissue was destroyed by this hemorrhage.	Marked fatty change.	No abnormality noted.	No abnormality.	

Ref No.	General Features of Case.	Duration of Failure.	Sinu-Auricular Node.	Auricular Muscle.	Ventricular Muscle.	A-V Connections.	Remarks.
75	F., 55. Chronic cardiac rheumatism with mitral and aortic fibrosis, and recent mitral endocarditis of doubtful character.	Probably of recent onset.	No abnormality.	Advanced fatty change.	Fatty change.	No abnormality.	Old glomerulitis noted in kidney.
82	M., 50. Chronic cardiac rheumatism, mitral stenosis.	Years.	No abnormality.	Advanced fatty change.	Fatty change only slight in left ventricle.	No abnormality.	—
95	F., 57. Chronic cardiac rheumatism; decrescent cardiosclerosis; recent endocarditis.	Days only.	Passive hyperæmia. Hypertrophy of A. to node.	Definite fatty change.	Definite fatty change. Cloudy swelling also noted in L.V. wall. Inflammatory foci, especially in I–V septum.	One large lymphocytic focus in early part of bundle.	—
102	F., 5. Chronic cardiac rheumatism; acute terminal carditis; chronic interstitial nephritis.	Months at least.	Normal; right auricle cavity closely approaches it.	Advanced fatty change in left auricle. Definite fatty change in right auricle.	Definite fatty change in both ventricles.	Normal.	—
156	F., 47. Chronic	Weeks.	Relative decrease of	Great variation	Slight fatty	Chronic obliter-	

156	F., 47. Chronic cardiac rheumatism. Terminal influenza with bronchopneumonia.	Weeks.	Relative decrease of muscle fibres, which also show some fatty change and hypertrophy. Artery to node shows hypermyotrophy. Capillaries and arterioles show mild endothelial reaction.	Great variation in size of fibres. Extremely advanced fatty change.	Slight fatty change.	Chronic obliterative reaction in artery to node.	—
107	F., 34. Chronic cardiac rheumamatism with mitral and tricuspid stenosis.	Years.	No abnormality.	Fibres vary much in size.	Slight fatty change.	Arterial hypertrophy. Slight fatty change in muscle fibres.	—
108	M., 29. Chronic cardiac rheumatism, sudden death.	Months at least.	No abnormality, except doubtful increase of connective tissue at lower pole.	Advanced fatty change.	Fatty change, advanced in places.	Obliterative inflammatory changes in vessels to node.	—
110	M 17. Acute rheumatic carditis terminating cardiac rheumatism.	About a year ?	Nil, except acute proliferative reaction in artery to node.	Fatty change, advanced in places.	No gross fatty changes. Many inflammatory foci.	Acute obliterative changes in arterioles.	—
			(*b*) Partial Examinations.				
67	F., 21. Chronic cardiac rheumatism. Very large heart, mitral fibrosis.	Over a year.	Not examined.	Advanced fatty change. Connective tissue increased.	Definite fatty change.	Not examined.	—
38	M., 59. Cardiosclerosis with hyperpiesis and terminal streptococcic endocarditis.	Weeks or months.	Not examined.	Advanced fatty change.	No gross changes.	Not examined.	—

Ref. No.	General Features of Case.	Duration of Failure.	Sinu-Auricular Node.	Auricular Muscle.	Ventricular Muscle.	A-V Connections.	Remarks.
68	F., 48. Chronic cardiac rheumatism with mitral stenosis.	5–10 years.	Not examined.	Advanced fatty change. One or two submiliary nodules.	No gross changes.	Not examined.	—
46	M., 5. Fulminating rheumatic carditis.	Days.	Partially examined; some inflammatory infiltration.	Saturated with inflammatory reaction.	Widespread and intense inflammatory reactions.	Partially examined. Some inflammatory reaction.	—
100	F., 31. Chronic cardiac rheumatism, with mitral stenosis; terminal endocarditis.	More than a year.	Not examined.	Cloudy swelling and segmentation.	Cloudy swelling and segmentation.	Not examined.	—
69	F., 41. Chronic cardiac rheumatism with mitral stenosis.	More than a year.	Not examined.	Advanced fatty change.	Definite fatty change.	Not examined.	—
57	M., 50. Alcoholic degeneration of myocardium.	Months.	Partially examined. No change.	Advanced fatty change.	Definite fatty change.	Not examined.	—
64	Child dying of diphtheria.	Days.	Not examined.	Advanced fatty change and cloudy swelling.	Definite fatty change and cloudy swelling.	Not examined.	—
66	M., 48. Chronic cardiac rheumatism, mitral stenosis, etc.	Years.	Not examined.	Advanced fatty change, etc.	Slight or definite fatty change.	Not examined.	—

difference between partial and complete examination is that in the latter serial sections were cut of the sinu-auricular node and the auriculo-ventricular connections, whereas in the partial examinations these series were omitted.

In only one case of auricular failure that was fully investigated was there any lesion of the sinu-auricular node that could conceivably have put an end to its effective action; and of that case, even, it is as true as it is of the others that significant degenerative changes were scattered freely throughout the auricular walls. Further, it is not unreasonable to think that the hemorrhage that had destroyed a part of the sinu-auricular node was a consequence, and not a cause, of auricular failure. Apart from this one instance the node was entirely free from gross morbid change.

On the other hand, in every case of auricular failure the musculature of the auricular walls had reached a pitch of deterioration so extreme that it was demonstrable by routine histological methods. The fatty change that has been accepted as the readiest index of that deterioration was most advanced in the parts of the auricular muscle that lay just under the endocardium. In some hearts the areas lying near the tip of the appendices displayed this most clearly, in others the change was at its maximum in the main auricular walls. Shrinkage of the decadent muscle cells brings into unnatural prominence the delicate interstitial stroma of the auricular walls. In the walls of the ventricles, on the other hand, changes such as these are barely discernible, and only in small and scattered patches.

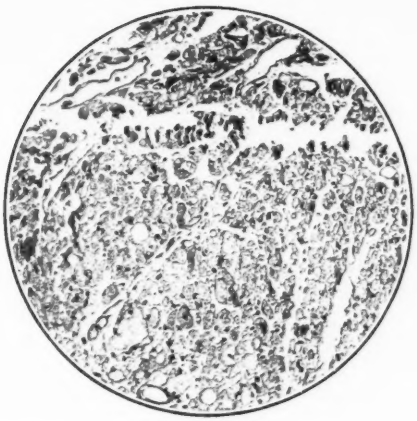
This contrast between the decadence of the auricles and the integrity of the ventricular walls is brought into greater relief if it is compared with two other sets of facts; first, with the absence of any such marked contrast from the

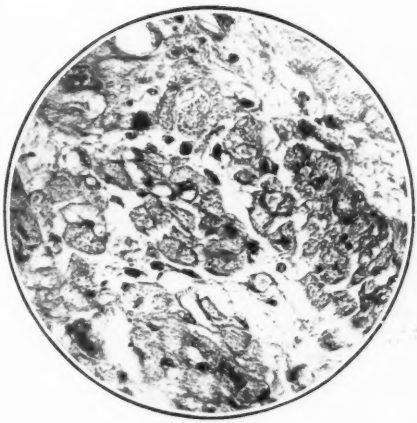
26 "control" specimens[1] alluded to above, and second, with the freedom of the auricular walls from inflammatory lesions in cases of acute cardiac infection. To each of these generalisations there are exceptions. For example, records 58, 61 and 76 were of cases of chronic cardiac rheumatism, with predominating mitral lesions but no signs of auricular fibrillation in life. In these the auricular muscle was grossly degenerate, much more so than that of the ventricles, showing that in such cases this contrast becomes apparent even before the arrhythmia that is characteristic of auricular failure sets in. Again, record No. 46 is that of a case of acute rheumatic carditis in which the inflammatory lesions of the auricular muscle surpassed those of the ventricles both in diffuseness and in intensity. Yet both of these exceptions do but support those generalisations which we feel justified in drawing from our observations of the histological changes that accompany auricular failure; first, that it is the whole auricular wall that "fails" and not the sinu-auricular node; and, second, that failure is usually the outcome of deterioration of the muscle cell itself. How profound that deterioration may be is well seen in the accompanying photomicrographs (Figs. 11 and 12).

Prognosis and Treatment.

Auricular failure, being practically always associated with advanced organic change, brings into any case of heart disease an element of gravity; first, because it is itself strong evidence of serious myocardial deterioration; second, because its rapid irregular rhythm lays on the ventricle a new burden. It is on this second fact that the prognosis of auricular failure largely turns. If the ventricles are already

[1] It is true that the texture of the auricular wall is such that it is easier to detect these changes in it than in the denser ventricular muscle, but this difference by no means explains away the whole of the contrast to which we refer.





FIGS. 11 and 12.

Photomicrographs showing myocardial degeneration in the auricular wall in auricular failure. The upper is a low-power and the lower a high-power photograph.

(*Blocks from "Rheumatic Heart Disease," kindly lent by Messrs. John Wright and Sons Ltd.*)

diseased and ineffective when the auricular functions fail, it is unlikely that treatment will afford a large measure of relief. Some relief there will be if, under appropriate treatment, the auricles are induced to beat regularly, or, failing this, fewer stimuli are allowed to pass through into the ventricles ; and if the ventricles are still in fair condition, the control of auricular failure furnishes for therapeutics one of the most striking opportunities of triumph in the whole field of medicine.

We have many examples of persons whose cardiac symptoms were so far allayed and kept in check by these means, that they were enabled to carry on their business for many years in comparative comfort. But others are insusceptible of relief ; and this is partly because, in a few instances, the drugs of which we are wont to expect a good deal prove ineffective, but more often because the ventricles are so far gone in disease that even when they have been relieved of the burden thrown upon them by failure of the auricular functions they cannot do their work effectively.

Many other elements may be introduced into a discussion of the prognostic significance of auricular failure ; but we wish to limit ourselves here to a statement of our belief that there are three principal sets of facts to be considered if we are to forecast truly the expectations of a case of cardiac disease in which the syndrome of auricular fibrillation is discovered. Those three are, first, the nature of the underlying lesion, whether it is transient or progressive, and, second, the state of the ventricles as far as this can be ascertained ; third, the behaviour of the fibrillating auricle under the influence of the appropriate drugs.

In practically all cases of cardiac disease presenting signs of auricular failure the first indication is to reduce the cardiac speed and thus to lengthen diastole. This indication is met by the use of digitalis. For patients whose symptoms

are only moderately severe moderate doses will suffice. For example, people who are not so ill but that they can attend an out-patient department take 15 minims of the tincture, or a pill containing one or two grains of the powdered leaf, every four hours. But it is for those who, when they first come under treatment, are urgently and even dangerously ill, that we would specially urge the use of digitalis in massive doses. Two or three years ago Eggleston of New York began to give the drug in a new way for cases of this kind. He showed that it was safe to give the dose which was just below toxicity in a single mass. The equation relating that dose to the patient's body weight is such that for an adult of average weight the dose would be three or four drachms of the tincture.

We have secured really remarkable results from a modification of this method, followed for two or three years; results paralleled in the experience of others who, independently of ourselves, adopted closely similar modifications of the Eggleston method. Our plan is to get three drachms taken in the first twenty-four hours. The accompanying chart (Fig. 13) shows how this plan brought down the heart rate in one case of this kind. We have seen lives prolonged for a year or more in at least a dozen instances, that would indubitably have been lost under any less thorough treatment. It is wonderful to see the relief afforded to the patient; and this, with reduction of the heart and pulse rates, and increase in the output of urine, may be confidently looked for in a majority of the rheumatic cases. We split our dose up in various ways: a good method is to give an initial dose of one drachm of the tincture, and to follow this by half-drachm doses at intervals of four hours till three drachms in all have been taken. The drug is stopped for twenty-four hours or more, and then resumed at a rate of fifteen minims every four

hours. This kind of plan has been followed in somewhere about fifty cases, and we have never yet seen toxic symptoms. More than once we have seen vomiting actually checked by digitalis in massive doses. We wish to lay the greatest possible emphasis on this, that the syndrome of auricular failure constitutes an absolute indication for the use of digitalis; that no other drug will take its place, and that no other syndrome is anything like so sure of relief from its use.

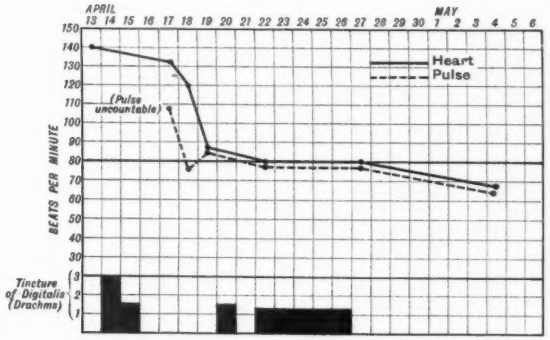


FIG. 13.

Chart illustrating response of heart rate and pulse rate to treatment by massive doses of digitalis.

(Block from "Rheumatic Heart Disease," kindly lent by Messrs. John Wright and Sons Ltd.)

The other drug that may achieve striking success in this syndrome, quinidine, has an action totally different from that of digitalis. Whereas digitalis reduces the cardiac speed without pretending to restore the normal rhythm, quinidine aims at an actual restoration of the normal rhythm. Our own experience of quinidine, based on more than thirty cases, shows that in about one-third it has no effect at all; in another third it has a fleeting effect on the rhythm; in another third it achieves a more lasting success. But

there are several reservations to be added to this statement. In the first place, most of the successes are not really permanent; they only last a few days or weeks, and then the abnormal rhythm returns. Second, in a few instances a fibrillation rhythm turns over to normal either spontaneously or under the influence of digitalis, and it is possible that some of the quinidine successes would have proved amenable to other plans of treatment. Then, again, the use of quinidine is not without risk. In one instance we have known sudden death occur during its administration. Other cases of the same kind are on record, as well as a number in which revival of auricular systole by quinidine was followed by embolism from the disturbance of clot which had settled down in the paralysed auricular appendix. This drug should therefore be used only for the most carefully selected cases, and under close supervision, the patient being kept in bed meanwhile. If it is decided to give it, the plan followed by one of us (C. F. C.) is to administer a single dose of 5 grains of the sulphate, to see if the patient has an idiosyncrasy for the drug. If not, it is given in 5 grain doses, in pill or cachet, every four hours till the normal rhythm is restored. It is worth going on for four days to a week before abandoning the attempt as fruitless, but as a rule if quinidine is going to cure it does so at once. The reversion from the abnormal to the normal rhythm takes place abruptly and without any half-way stages. To maintain the normal rhythm it is well to give quinidine, 5 or 10 grains daily, for some long time.

The plan followed by the other of us (C. E. K. H.) differs in details only from the above. A single dose of 0·4 gramme (6 grains) is given on the first day, and if this is followed by no untoward results a similar dose is given every two hours, for five doses per day, this being continued for five days if the normal rhythm is not recalled in a shorter time.

Physiological Considerations.

These therapeutic procedures gain much in interest as well as in reliability because they are founded on a scientific basis, an application of pharmacology to the relief of diseased functions. As the result of work done by Lewis and his co-workers in London we now know the mechanism of the conditions known as auricular flutter and fibrillation. If a complete circle of muscle be stimulated to contract by an induction shock at a certain spot a wave of contraction starting from that point will pass round each side of the ring until it "meets itself" at the reverse side of the ring, when the contraction ceases. If, however, this ring of muscle has been made to contract by rapidly repeated induction shocks just before the experiment, it is found that during the partial recovery of refractoriness the wave of contraction may be stopped from passing up one side of the ring while it passes up the other side. This wave will then pass completely round the ring, and when it reaches its starting-point it will, provided the muscle has sufficiently recovered from its last contraction, start again round the ring, and so on indefinitely. The only condition necessary for the continuance of this phenomenon is that the time of partial recovery from the refractory period must be a little less than the time taken for the contraction to pass round the ring of muscle; in other words, there must be a gap between the muscle that is in an absolutely refractory state and the advancing wave of contraction. The time taken to complete the circuit depends chiefly upon the length of the path round the ring; the rate of the contraction wave and the length of the refractory period being more or less constant. Lewis has found that a similar condition of "circus movement" occurs in experimentally induced flutter and fibrillation in dogs, and there

is no doubt that the conditions in man are caused by the same mechanism.

The difference between fibrillation and flutter is mostly in the length of the path. In flutter the path is longer and the gap between the advancing wave of contraction and the refractory path is wide, and there is no hindrance to the path of the wave round the ring, so each circuit takes the same time, *i.e.* 1/300 of a second; therefore, in auricular flutter the auricle contracts 300 times a minute, the wave of contraction passing from the ring of muscle out to the rest of the auricular fibres. In fibrillation the path is shorter and the gap is narrower; consequently the muscle is more disinclined to contract than in flutter; in other words, the partial refractoriness is nearer the absolute. The wave of contraction, therefore, finds difficulty in passing round the ring of muscle, and has to discover a path for itself among fibres of which some are impassable, while others can be passed only with difficulty. No two circuits are composed of the same fibres, and the time taken to complete the circuit is not constant. For this reason the auricular rhythm in fibrillation is irregular, and since it takes about 1/450 of a second to complete the circuit, there are as many as 450 circuits of the ring of muscle per minute. The spread of contraction into the auricular muscle is irregular and much more rapid, some of the muscle is refractory, and there is no co-ordinate conduction from bundle to bundle; hence there is no co-ordinate contraction of the auricles, but the muscle is continuously quivering from the irregular contractions of individual bundles of fibres, giving the muscle the appearance of a fibrillary tremor like that met with in progressive muscular atrophy. Lewis has shown that in flutter the ring of muscle which is acting in this way is the muscle surrounding the apertures of the two venæ cavæ, while in fibrillation the wave of contraction

circles round one of the veins. In auricular flutter the ventricle does not respond to every auricular beat, as heart block of varying grade may be present, thus acting as a protecting mechanism; for the heart would rapidly fail if the ventricles tried to contract at a rate of 300 per minute. Similarly in fibrillation the auriculo-ventricular connections refuse transmission to a majority of the stimuli coming down from the auricles. It is in a strengthening of this inhibitive action of the auriculo-ventricular connections that the therapeutic value of digitalis lies.

Digitalis used in fairly large doses has a depressant action on the junctional tissues between the auricle and ventricle. It delays or decreases conductivity of the bundle, thereby preventing a large number of the stimuli from the fibrillating auricle reaching the ventricle. Hence the ventricle is slowed.

Its action on the fibrillating auricle is, as a rule, to quicken the rate of the contraction wave and to shorten the refractory period, consequently it does not alter the auricular rhythm, nor does it tend to stop the fibrillation. So that although it markedly improves the condition of the patient, one must not expect it to cure the fibrillation.

Quinidine acts in the opposite way; it tends to increase the ventricular rate, but on the auricles it has two actions, (1) it slows the rate of contraction, (2) it lengthens the refractory period. Sometimes one predominates, at others the other is the more marked. If its chief action is to lengthen the refractory period, the advancing wave of contraction round the ring of muscle catches up the refractory muscle, the gap is obliterated, the circus movement comes to an end and normal rhythm recommences. If the slowing of the rate of contraction predominates the time taken to complete the circuit is lengthened, the gap is unaltered and the fibrillation continues. This is the reason why quinidine sometimes stops fibrillation, and at others appears to have no such action.

A FEW SUGGESTIONS IN GENERAL SURGERY.[1]

BY

C. F. WALTERS, F.R.C.S.,

Surgeon to the Bristol Royal Infirmary.

PRESSURE FORCEPS TECHNIQUE.

MY first suggestion, one on general technique, proven this seven years, is based on a single page article of some ten years ago that I have been unable to unearth again, in which is described a method of tying ligatures by means of artery forceps. On this one has developed a technique by which all manœuvres of ligaturing and suturing are done by means of pressure forceps, so that one may perhaps describe it as " The Pressure Forceps Technique."

One has always seen in the so-called " spoon and fork " operation on bone, brain and joint an element of risk introduced by hand ligaturing, and sometimes one has seen the operation still further jeopardised by hand suturing when gloves are so readily torn, whereas by means of the Pressure Forceps Technique the surgeon's hands approach no nearer to the wound at any period of the operation than the distal end of the instrument in use. This applies also to the house surgeon and dressers. So that as far as the surgeon is concerned, from an aseptic point of view, the chain is complete, and bone, brain and joint operations can be embarked on with complete confidence.

This is the most important feature of this method, but there are others.

Accessibility is improved ; it is easy to ligature at a depth by sight when hand ligature must of necessity obscure the view, and is sometimes impossible.

[1] Read before a Meeting of the Bristol Medico-Chirurgical Society on May 14th, 1924.

While this method is best shown by cinema, with the written word one is confined to the following diagrams:—

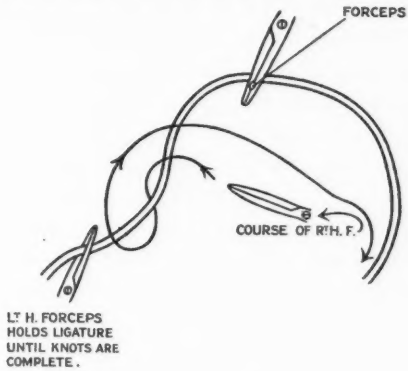


FIG. 1.

When second movement of right hand forceps is complete it has seized right end of ligature, it then passes to the right and the left to the left.

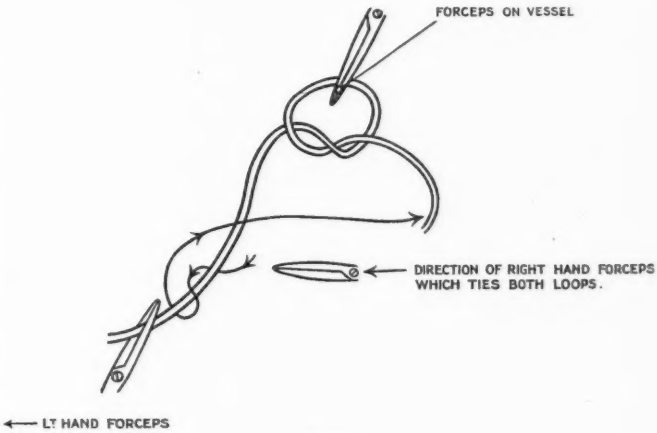


FIG. 2.

After this movement of the right-hand forceps is complete, and it has seized right end of ligature, the left-hand forceps passes to the right and the right-hand forceps to the left to complete the first loop.

Facility is greater; one can readily tie a ligature with one and a half inches at one end and a quarter of an inch at the other.

Rapidity is increased, forceps ligature being at least half as fast again as by hand.

Economy is a marked feature; it is possible to tie seven or eight times as many ligatures with the same length of catgut by forceps as by hand, and when the yearly hospital bills for catgut are so large it is worth considering.

The complete method is applicable to all surface operations and the tying and suturing to many deep ones.

Lastly, the mental comfort of having thus dealt with bone, brain and joint, in fact any operation where a perfect aseptic technique is obtained, is alone in my opinion well worth the while.

Abdominal Drainage.

Next I would suggest that a dental rubber dam drain down to but not through the peritoneum and left in position for six or seven days is, *so to speak*, an antidote to incisional herniæ in septic appendix wounds in particular, and septic abdominal wounds generally, by which I mean wounds inevitably soiled owing to the presence of free pus in the peritoneum.

My clinical impression—and one knows what such are worth—after utilising this method for the last three years was that suppuration was prevented or diminished, a sounder wound and healing by first intention more frequently obtained where free pus was present in the abdomen.

In an attempt to prove this I took two periods, the past three years, during which I have used a dental rubber dam drain, and the three years previous to that, during which no drain was introduced into the appendix wound itself.

In some 300 cases of appendicitis on which I had operated

during both periods 41 cases during the first where no drain was used were recorded as having free pus in the abdomen, and 54 in the second period, when rubber dam was utilised.

Despite many personal visits to their houses in an attempt to see the wounds, I was able to see only 25 out of the 95 in which the abdomen contained free pus at the time of operation.

Of these, 8 were in the first period without drain and 17 in the second period with it. Of the 8 seen without drain four had well-marked herniæ, of the 17 with drain only one had a hernia. These figures are far too small to prove anything, but I would suggest that where a wound is necessarily infected by the presence of free pus in the abdomen a dental rubber dam drain down to but not through the peritoneum and left *in situ* for six or seven days will in such cases tend to lessen the incidence of incisional hernia.

Of my next two suggestions one carries no proof for lack of material and the other needs more experience, but I venture to put them forward in the hope that if they are worthy of consideration others may test their worth or lack of it.

Exteriorisation.

First, as to the thorax. Those who have delved much in thoracic surgery, and for my own part I have tried most known and some unknown methods, will agree as to the difficulty in closing chronic empyema cavities, whether it be by Estlander, Schede, decortication, discission, sub-costal pleural freeing, and what not.

The difficulties of pre-operative sterilisation, the failure of lung to remain distended that expanded freely at operation, recurrent suppuration months after an apparently successful healed decortication—sometimes the complete failure of the procedure—and on one occasion, in my own experience, a fatal ending, may bring one's efforts to naught.

For the larger cavities I have nothing new to offer; but for the smaller, those of fist or sausage size and shape which sometimes fail to close despite apparently adequate drainage, I would suggest what I have called *Exteriorisation*, though my experience is based on one case only—that of a young man with a cavity of some months' standing due to collapse of the left lower lobe following empyema.

For weeks I attempted to sterilise this as a preliminary to decortication, and failed, and in despair turned up the whole anterior wall of the cavity, sub-periosteally excising sections of three ribs, sewing the flap to the chest wall above, and packing with Eusol the completely-exposed cavity. To my astonishment it rapidly granulated, the flap tore out its fixing stitches, replaced itself and healed.

Eighteen months later I saw him; he was doing his usual work with ease, and although the upper lobe only was functioning the wound had remained soundly healed.

The Dural Flap.

Not infrequently one is asked by one's medical colleagues to do a decompression for symptoms of cerebral tumour, and one has not failed to note the anxiety of the physician that the surgeon should stop at the dura, fearing—not unjustly—cerebral hernia if it be opened.

Sometimes one has respected this wish and sometimes disregarded it with regret, but if the dura can be opened and readily closed without fear of hernia it may relieve the physician of his fears and justify the curiosity of the surgeon.

One is aware that cerebral tension can be diminished by ventricular puncture, though this is not without danger and may fail, and one has found that hypertonic saline will deplete the brain of fluid; but I would suggest what may be described as the *Dural flap* as a means by which cerebral hernia can be prevented, the dura readily restored with more

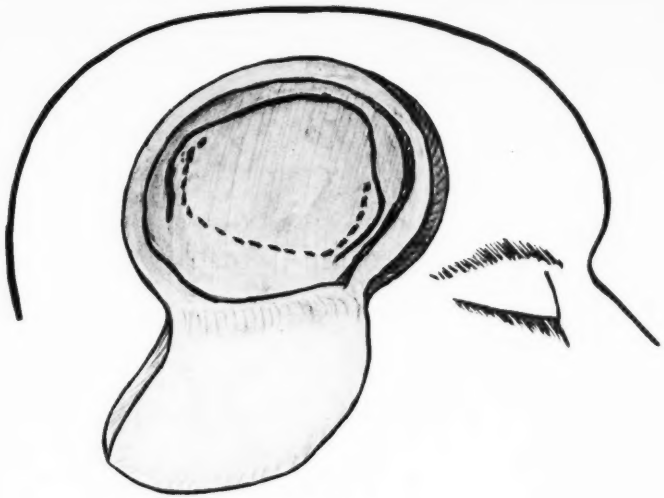


FIG. 3.
Superficial Flap Continuous Line—Deep Dotted.

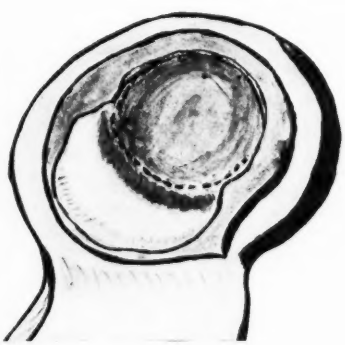


FIG. 4.
Superficial Flap Raised.

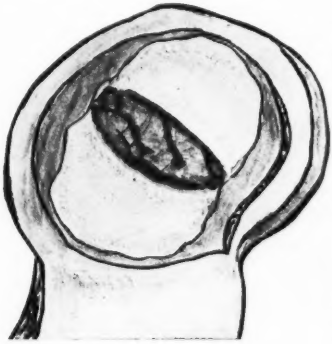


FIG. 5.
Both Flaps-of-Dura Everted.

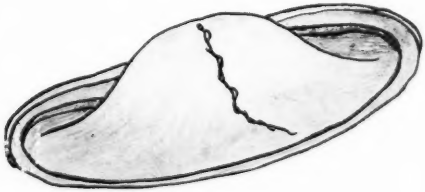


FIG. 6.
Flaps Closed.

ample decompression, the physician comforted and the surgeon almost satisfied.

Method of Flap Cutting.

The dura having been exposed with a very sharp broad-bellied scalpel, the flat surface of which is held almost parallel to the dura approaching it at a very slight angle, the outer surface of the membrane is raised following the outline of the flap (Fig. 3) for about a sixteenth of an inch. This flap is then seized with finely-toothed forceps and pulled on, when the dura will be found to split readily into two layers attached to one another by fine fibrous processes.

If the knife is held as before mentioned and drawn across these fine processes the splitting can be fairly rapidly proceeded with, and sometimes further facilitated by stripping with gauze held in forceps.

The first flap having been raised (Fig. 4), of such a shape as the diagram shows, the deeper layer of the dura is then divided as indicated (Fig. 5), when sufficient redundant membrane will be found not only to readily cover the protruding brain tissue, but to allow also an increased space for decompression (Fig. 6).

Since this method occurred to me some three months ago I have had four opportunities of testing it, two on children and two on adults.

I may say at once it fails in the child, its dura is too thin for splitting, but fortunately is more elastic and more readily closed than that of the adult. In the adults it proved easy and was successful.

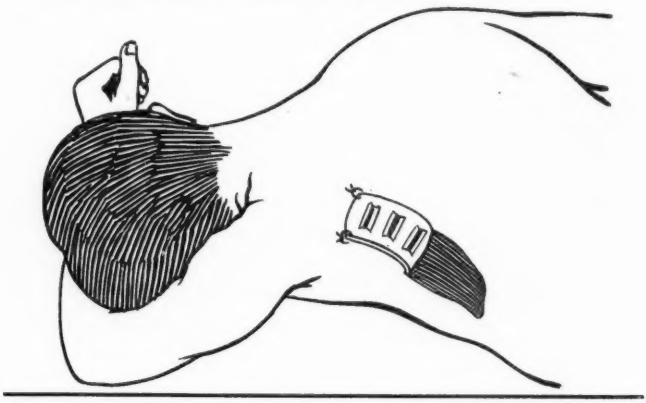
One adult died a month after operation, and the portion of dura in which the flap had been cut was obtained and the finding somewhat unexpected. The under surface of the superficial flap, that with a raw surface, was perfectly smooth, while that of the lower flap, the true inner dural

surface, was slightly rough, but nowhere was either flap adherent to the brain.

RECUMBENT EXERCISES DURING CONVALESCENCE.

Lastly, as to convalescence—and this should apply not only to surgical but to medical cases also. It becomes the practice in surgery more and more to lessen the time, following operation, during which a patient is what one may call bed-ridden.

Frequently the simple appendix patient is on his or her feet at the end of a week ; some surgeons get them up still earlier, and some would, if they could, walk them off the operation table ; but whether it be one, two, or three weeks that a patient must be kept in bed muscular power and tone is diminished from disuse, and after longer periods the first progress from bed to chair is only too often a tottering effort needing much external support.



This I suggest is unnecessary and can be largely eliminated by practising, as I have for some period, what I have called *Recumbent Exercises*, by which when the patient is allowed to get up he can literally, though unable to manage the middle of the quotation, arise and walk.

The most gentle manner in which these exercises can be applied to the aged and debilitated is in the form of deep breathing, five deep breaths after each meal, increasing by one or more daily at each period, and so improving oxygenation and tending to prevent hypostatic congestion and possibly other complications. The exercises in their complete form bring into play every important body muscle, and in the increasing doses mentioned not only maintain muscular tone but actually in the fat and indolent better their pre-operative muscular condition and, moreover, give them something to think about.

The whole series can be done in the supine position, and although suitable exercises will at once occur to any medical man, I can illustrate those that I use by the following rough diagrams (Fig. 8).

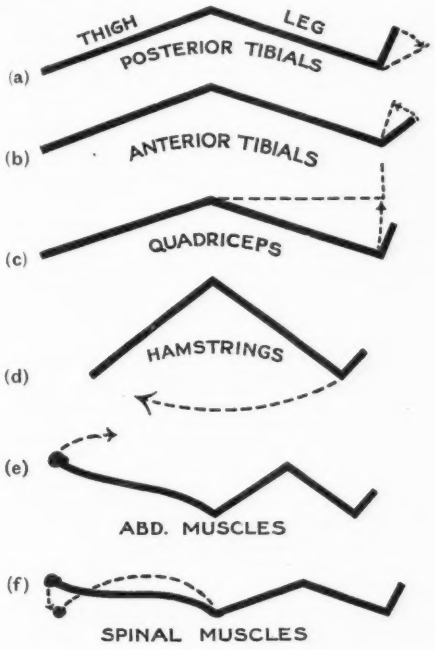


FIG. 8.

Reviews of Books.

Organic Substances, Sera and Vaccines. By D. W. CARMALT JONES, M.D. (Oxon.), F.R.C.P. (Lond.). Pp. viii., 393. London: William Heinemann. 1924. Price 15s. net.—This book appears to have been designed to meet the want of the general practitioner who desires a small volume which will give advice on the uses of remedies acting in a biological rather than in a chemical sense, both terms being applied loosely. The author deals with endocrine substances, specific and other sera, proteins, vaccines and toxins. The ground to be covered is wide, and much skill was necessary to limit the result to a small single volume. A few points deserve attention. Misprints are rather numerous, especially in the spelling of the names of persons. The opening paragraph to chap. iii. requires revision. Loewi's test as described would be positive in about 90 per cent. of normal subjects; in this test the dilatation of the pupil following the second application of adrenaline constitutes a positive result. The footnote on p. 182 appears obscure. In the rationale of normal serum therapy the explanation given is probably incorrect: a few cubic centimetres of normal serum are hardly likely to contain sufficient coagulative adjuvants to give such dramatic results. The addition of anti-anaphylactic substances, including normal saline, when serum is to be administered intravenously might be stressed. The importance of haste in the transmission of dysentery and other stools to the laboratory requires much more emphasis. On anthrax the author's pessimism regarding the utility of treatment other than specific serotherapy is hardly justified. In his review of vaccine treatment a more optimistic note might have resulted from extracts from more modern articles: thus in the treatment of typhoid fever and pneumonia a good deal of recent work appears to confirm the opinion of the utility of vaccines. Lastly, the index might be enlarged and improved by cross references. These, however, are minor points, and should be remedied in the second edition. On the other scale the author has treated his subjects carefully and cautiously; undue optimism is repressed, a point of some importance in this branch of therapeutics. The section on the collection and transmission of specimens for diagnosis is good, and might be lengthened with profit both to practitioner and pathologist. The work is well up to date; recent gains are noted with

caution ; one omission, perhaps intentional, is the phenomenon of Twort-Herelle. The whole book is good. The author has succeeded in his attempt, if the reviewer's opinion of his aim is correct. The work should be of marked practical value.

Venereal Disease. Its Preventive Symptoms and Treatment. By WANSEY BAYLEY, M.C. Pp. xvi., 176. London : J. & A. Churchill. 1924. Price 7s. 6d. net.—In this little book the author has placed before his readers an enthusiastic exposition of the important question of venereal disease. It contains a full and fascinating description of the essentials for the prevention, diagnosis and treatment of venereal disease presented to the practitioner in a clear and simple manner. That this edition is the outcome of honest hard work is obvious. Whilst it omits certain tests which are considered unreliable, such as the complement fixation and cultural tests for gonorrhœa, it is most up to date in its reference to such recent work as Professor Levaditi's on bismuth and Professor Fourneau's on stovassol. The methods of prevention advocated are sound and practical.

Gonorrhœa. By DAVID THOMSON, O.B.E., M.B., Ch.B. Edin., D.P.H. Camb. Pp. xiv., 519. London : Henry Frowde and Hodder and Stoughton. 1923. Price £2 2s. 0d. net.—This volume is the outcome of seven years' work by the writer on the bacteriology of gonorrhœa. He has given the most complete work that has yet appeared on the subject from any British author. In the main the book is a treatise on the bacteriology of the disease, and more than half of the work is devoted to laboratory methods. His chapters on the cultivation of the gonococcus, its toxins and associates, are excellent. In a later chapter he deals very fully with immunisation by means of vaccine therapy, and favours the use of specific vaccines. Another chapter is devoted to a full account of his work on detoxicated vaccines, and he answers his critics clearly and convincingly. He has sought the assistance of first-class authorities in writing the chapter on vaccine treatment, as well as in those on other forms of modern treatment, including abortive methods. These chapters are eminently practical, and may appeal to a larger circle of practitioners and students than the bacteriological portions of the book. The illustrations, especially the coloured plates, are excellent. The volume provides both attractive and instructive reading, and must have entailed an enormous amount of honest work.

Notes on the Composition of Scientific Papers.—By The Right Hon. Sir T. CLIFFORD ALLBUTT. P.C., K.C.B., M.A., M.D., etc. Pp. xii., 192. London : Macmillan & Co. Ltd. 1923. Price 6s.—Those who were already familiar with Sir

Clifford Allbutt's little book of urbane and polished admonitions, in its earlier form, will not be surprised that a new edition has been called for. It is not only that it is alone in its particular field. There is also a charm of style which is in itself the best possible medium for the lesson that its author wishes to teach. Both by example and by precept, however, he sets such a standard of excellence as may well be the despair of lesser mortals, and one might be tempted to remark cynically that not the least merit of the book is that it should serve to discourage persons from the attempt to compose scientific papers. It is an unfortunate fact that many papers are written which ought not to have been written, while many are left unwritten which ought to have been written. We believe that the reading of this book would inspire many to avoid both kinds of error, and for this as well as for other good reasons we commend it to everyone engaged in the struggle against disease.

THE FARR FUND.

THIS fund, which has been steadily increasing, now totals £129 5s. 0d. The President hopes to raise this to £150. There must still be members of the Society who have not yet contributed, but will like to do so. If they would send in their subscriptions at an early date it would help materially, as to be of real good the fund cannot be kept open much longer.

†

IN MEMORIAM.

Lemuel Matthews Griffiths, M.R.C.S., L.R.C.P.

THE Bristol Medico-Chirurgical Society mourns the loss of Lemuel Matthews Griffiths, who died on September 22nd, in his eightieth year, an Ex-President and one of its earliest and most active members. With this issue already in the press it is quite impossible to do justice to his memory or give adequate expression to the well-earned gratitude of his medical confrères in Bristol for the many years of splendid service rendered towards founding, establishing and developing both our Medical Library and also this Journal.

The recital of even this one aspect of the life's activities of one so beloved by all who knew him demands more time than is now available. But unknown to most of his contemporaries, he also devoted much of his time and energies to sociological work in the poorer districts of our City, while he attained a world-wide reputation amongst Shakespearean scholars.

Our readers will be glad to hear that his close friend, Dr. Alexander Smith, has kindly undertaken to contribute a full obituary notice in our next issue. Nevertheless, we cannot refrain from noting the passing hence of one who has laid the whole Medical profession and more particularly the Society and its Journal under deep and lasting indebtedness.

Editorial Notes.

The University accepts our Society's Library.

THE Medical Library, of which the Medico-Chirurgical Society is justly proud, was offered to and has been accepted by the Council of the University on conditions which preserve all the privileges of existing members and which are to be extended to all future members of the Society. The Council of the University, however, has shown their appreciation of this gift by providing a noble library with ample accommodation for its future expansion, as well as a large room for current periodicals and, furthermore, a room set aside for the use of the Society's members. We have no reason to doubt that the University, if not now, at any rate in the long run, will give to our Society as much if not more than they have received, and that our members will benefit very greatly from the transaction.

The Society's Journal has been a potent factor in the building up of this Library in the past, and should be no less so in its further development. The interests subserved by the Medical Faculty of the University and by this Journal are now more closely interwoven, and as a consequence we trust the sphere of our Journal's utility will be enhanced and widened.

We rejoice that Mr. L. M. Griffiths lived to know of this happy consummation of his great work for the Society, its Library and its Journal.

The Colston Research Society.

A GRANT from the Colston Research Society has made it possible to publish the clinical and pathological research on Auricular Failure by Drs. Carey Coombs and Herapath, and reproduce many of their original illustrations. It is not our purpose here to touch on the value of this contribution, though representing results of scientific investigation which advances our knowledge and solves some of the inherent problems of this aspect of cardiology, but we do feel that the Colston Research Society has earned the thanks of Medical Science for the encouragement and assistance afforded to such research and to our own Society by enabling us to bring the results before our readers in a complete form.

The Library of the Bristol Medico-Chirurgical Society.

The following donations have been received since the publication of the List in July, 1924.

	October, 1924.
Dental Board of the United Kingdom (1) ..	1 volume.
Mr. Hey Groves (2)	1 ,,
Medical Research Council (3)	2 volumes.
Surgeon-General of the United States Army (4)	2 ,,

Unbound periodicals have been received from Lieut.-Col. A. B. Prowse, M.D., F.R.C.S., and Mr. Hey Groves.

THE ONE HUNDRED AND TENTH LIST OF BOOKS.

Buchanan, A. ..*Midwifery Mechanics* 1924
Burridge, H. A. ..*An Introduction to Forensic Medicine* 1924
Child Life Investigations .. .. (3) 1924
Culpin, M. .. ..*The Nervous Patient* 1924
Fitzwilliam, D. C. L. *On the Breast* 1924
Growth of the Jaws, Normal and Abnormal, in Health and Disease (Five Lectures) .. .. (1) 1924

Haig, H. A.	*An Introduction to the Histology of Tumours*	1924
Hurst, A. F.	*Essays and Addresses on Digestive and Nervous Diseases, etc.*	1924
Index-Catalogue, Library of the Surgeon-General's Office, United States Army. ..	3rd Ser., Vol. IV. (4)	1923
McDonagh, J. E. R.	*The Nature of Disease*	1924
Martindale and Westcott.	*The Extra Pharmacopœia.* 18th Ed. Vol. I.	1924
Medical Department of the United States Army in the World War,	Vol. XI. .. .. (4)	1924
Muir, R.	*Text-Book of Pathology*	1924
Oehlecker, F. ..	*Tuberkulose der Knochen und Gelenke* .. (2)	1924
Pickerill, H. P. ..	*Facial Surgery*	1924
Report on the Nutrition of Miners and their Families	(3)	1924
Schmidt, P.	*The Theory and Practice of the Steinach Operation*	1924

TRANSACTIONS, REPORTS, JOURNALS, ETC.

American Ophthalmological Society, Transactions of the Vol. XXI.	1923
College of Physicians of Philadelphia, Transactions of the, 3rd Ser. Vol. XLV.	1923
Medical Officer of Health, Bristol, Annual Report of the	1923
University of Otago Medical School, Proceedings of the, No. 2 ..	1924

Local Medical Notes.

MARRIAGE.—On August 18th, at Crantock, Cornwall, the marriage took place of J. A. Nixon, C.M.G., M.D., F.R.C.P., Professor of Medicine, University of Bristol, and Physician, Bristol Royal Infirmary, to Miss D. G. C. Walker, M.R.C.S., L.R.C.P.

Postgraduate Demonstrations of the University of Bristol are to be held at the Town Club, 59 Bath Road, Swindon, on Wednesdays, at 3.15 p.m., viz.: October 29th, "Hepatic Insufficiency, Diagnosis, etc." (Dr. A. T. Todd); November 5th, "Inter-relations of Internal Secretion Glands" (Professor F. H. Edgeworth, M.D.); November 12th, "Tonsils and Adenoids and Ear Discharges" (Dr. A. J. Wright); November 19th, "Care and Treatment of Children's Mouths" (Mr. L. E. Claremont, M.R.C.S., L.D.S.); November 26th, "Defective Dietary as a Cause of Children's Disorders" (Dr. O. C. M. Davis); December 3rd, "Dyspepsia: some considerations for diagnosis and treatment" (Dr. Carey Coombs).

Dr. F. C. V. Thompson, Wroughton, Swindon, is the acting Hon. Sec. The fee is Two Guineas for the course.

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